Archana Singh · Indrakant K. Singh
*Editors*

# Molecular Aspects of Plant-Pathogen Interaction

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# Preface

Pathogen attack has been one of the chief constrains that reduce crop productivity worldwide. Plants have established sophisticated mechanisms to counter and acclimatize over these invading pathogens at physiological, biochemical as well as molecular levels. Due to severe crop losses by pathogen outbreak, it is mandatory to completely understand the resistance/defense mechanisms against pathogen and develop advanced tactics to improve biotic stress tolerance in crop plants.

We present this book with an objective to realize the plant defense against different pathogens better and to document fundamentals as well as recent findings. This book has an amalgamation of basic information about disease resistance along with current insights into plant-pathogen interaction. The book has 15 chapters to disseminate the most updated information and detailed overviews on the present knowledge on molecular aspects of plant responses and adaptation to biotic stresses. This book is an essential reading for researchers and professionals in plant pathology, cell biology, molecular biology and genetics. This is highly recommended for the ones who are involved in plant disease resistance and crop improvement and to all plant scientists and undergraduates.

Depending on their modes of nutrition, phytopathogens have been categorized as necrotrophs, biotrophs and hemibiotrophs. These pathogens can be bacterial and fungal and cause various diseases in plants. In addition, viruses are another important class of pathogens and are causal agents for many common plant diseases. Plants counter to pathogens by activating a cascade of genes, encoding different receptors, signaling and protective molecules. During biotic stress, first of all effector molecules i.e. pathogen-associated molecular patterns (PAMPs) are perceived by plant recognition receptors (PRRs), after which PRRs interact with additional trans-membrane proteins that act as signaling adapters or amplifiers to achieve full functionality and PAMP triggered immunity (PTI). Defense response by receptor-like protein is a complex strategy, characterized by specific interaction between disease resistance (*R*) genes of plants and corresponding avirulence (*avr*) genes of pathogen that induce effector-triggered immunity (ETI) through hypersensitive response.

The NBS-LRR genes are important class of resistance gene families and their products recognize factors secreted by pathogens, which activates downstream signaling pathways leading to defense. Mitogen-activated protein kinases (MAPKs), which are cell-signaling enzymes that also show vital functions in transmitting extracellular signals to the nucleus during biotic stress. To achieve defense against

pathogen, transcription factors such as WRKY transcription factors bind to plant-specific *cis*-regulatory elements and activate gene expression thereby inducing transcriptional reprogramming and proteomic alterations to coordinate the perception and activation of pathways specific to the type of pathogen in question. Mainly phytohormones, small RNAs and other factors regulate this change at transcript level and protein level. Amongst all the targets, the induction and accumulation of pathogenesis-related (PR) proteins and biosynthesis of secondary metabolites are an integral component of innate immune responses in plants during pathogen attack.

Overall this volume will convey an overview of plant-pathogen interactions and it is a must read to understand this process for the genetic improvement of crops for disease resistance.

We are obliged to the authors of various chapters of this book for writing their chapters methodically and with great responsibility. We are extremely thankful to Dr. Rama, Principal, Hans Raj College, University of Delhi and Dr. Ajay Arora, Principal, Deshbandhu College, University of Delhi for providing overall support for our research and academic pursuits. We would also like to convey our gratitude to Dr. V. K. Kawatra, Mr. P. K. Singh and Dr. Vijay Rani Rajpal for always motivating us. We appreciate the beautiful ambiance created by our little angels Saumya and Kimaya, which allowed us to work tirelessly and gave us all emotional support. We are grateful to our parents for their constant support and blessings. Last but not the least, our sincere thanks to the handling editors and publisher.

We are optimistic that this book will be effective in broadcasting the latest knowledge about the plant-pathogen interaction.

New Delhi, India

Archana Singh
Indrakant K. Singh

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**Dr. Archana Singh** is a Ph.D. in Plant Molecular Biology from the National Institute for Plant Genome Research (NIPGR). Since 2009, Dr. Archana has been working as an Assistant Professor in the Department of Botany, Hans Raj College, University of Delhi, where she teaches plant biotechnology, bioinformatics, genetics, cell biology and molecular biology. She has been awarded with Young Scientist Under Fast-Track Scheme (2013–2016) by Science and Engineering Research Board (SERB), Ministry of Science of Technology, Government of India. She has also been awarded with a prestigious Raman Post-Doctoral Fellowship by University Grant Commission, Ministry of Human Resources and Development, Government of India. Her quality research is evidenced by her publications in peer-reviewed journals of international repute like *Journal of Experimental Botany*, *Scientific Reports*, *Journal of Integrated Plant Biology*, *Journal of Asia-Pacific Entomology*, *Australasian Plant Pathology*, *Computational Biology and Chemistry* and *Plant Gene*. She has trained and supervised a few Junior Research Fellows and M.Sc. and B.Tech. students for their research projects. Over the years, she expanded her research interests from functional genomics of biotic stress to bioinformatics and proteomics.

**Dr. Indrakant K. Singh** is a Ph.D. in Zoology from the University of Delhi (DU). For last ten years, he has been working as an Assistant Professor in the Department of Zoology, Deshbandhu College, University of Delhi, where he teaches genetics, genomics, computation biology and immunology. He has been awarded with Young Scientist Award/Fellowship (2013–2016) by Science and Engineering Research Board (SERB), Ministry of Science of Technology, Government of India. He has also been awarded with a prestigious Raman Post-Doctoral Fellowship (2016–2017) by University Grant Commission, Ministry of Human Resources and Development, Government of India. He has published his quality research in peer-reviewed journals of international repute like *Journal of Experimental Botany*, *Scientific Reports*, *Journal of Biomedical Research*, *Journal of Asia-Pacific Entomology*, *Australasian Plant Pathology, Computational Biology and Chemistry* and *Plant Gene.* Presently, two Ph.D. scholars are working under his supervision. He has trained several B.Sc. and M.Sc. students for their research projects and now they are pursuing their doctoral research abroad. Over the years, he expanded his research interest from molecular basis of insect-plant interaction to functional genomics and proteomics.

# Abbreviations

| | |
|---|---|
| 12-OPDA | 12-oxophytodienoic acid |
| 24-EpiBL | 24-epibrassinolide |
| 28-HomoBL | 28-homobrassinolide |
| 2D-DIGE | Two dimensional differential gel electrophoresis |
| 2D-PAGE | Two dimensional polyacrylamide gel electrophoresis |
| 5-HPT | 5-hydroxytryptophan |
| AABL1 | AB15 like1 |
| ABA | Abscisic acid |
| ABC | ATP binding cassette |
| ACC | 1-aminocyclopropane-1-carboxylic aid |
| ACMV | African cassava mosaic virus |
| ACS6 | 1-aminocyclopropane-1-carboxylic acid synthase |
| AGO | Argonaute |
| ALS | Amyotrophic lateral sclerosis |
| AMV | Alfaalfa mosaic virus |
| AP2 | Apetala2 |
| APAF | Apoptotic protease activating factor |
| APX | Ascorbate peroxidase |
| ARID | A/T rich interaction domain |
| as-1 | Activation sequence 1 |
| At | *Agrobacterium tumefaciens* |
| AtAGO1 | *Arabidopsis* AGO1 |
| AtEFR | *Arabidiopsis thaliana* EF-TU receptor |
| AtFSL2 | *Arabidopsis thaliana* flagellin sensing 2 |
| ATRM | *Arabidopsis* transcriptional regulatory map |
| ATX1 | *Arabidopsis* homolog of trithorax |
| Aux | Auxin |
| Avr | Avirulence |
| BAK1 | BRI1 associated receptor kinase 1 |
| BAW | Beet armyworm |
| Bc | Botrytis cinerea |
| BECs | Blumeria effector candidates |
| BHNs | Broad host range necrotrophs |
| BIA | β-(Isoxazolin-5-on-2yl)-alanine |

| | |
|---|---|
| BIK1 | BRI1 associated kinase |
| BIN2 | Brassinosteroid insensitive 2 |
| BL | Brassinolide |
| BMAA | β-methylamino-L-alanine |
| BPH | Brown plant hopper |
| BRI1 | Brassinosteroid insensitive 1 |
| BRs | Brassinosteroids |
| BWMK1 | Blast and wounding activated map KINASE 1 |
| bZIP | Basic domain leucine zipper |
| CaBD | Calmodulin binding domain |
| CaM | Calmodulin |
| CARD | Caspase recruitment domain |
| Cas | CRISPR associated |
| CAT | Catalase |
| CBB | Coomassie brilliant blue |
| CBD | Chitin binding domain |
| CC | Coiled coil |
| CD | C-terminal common docking |
| cDNA | Complementary DNA |
| CDPK | Calcium dependent protein kinase |
| CEBiP | Chitin elicitor binding protein |
| CED4 | Cell death protein 4 |
| CERK1 | Chitin elicitor receptor kinase 1 |
| CESA | Cellulose synthase cataylic subunit |
| CGs | Cyogenic glycosides |
| ChIP | Chromatin immunoprecipitation |
| CHS | Chalcone synthase |
| CKs | Cytokinins |
| CMV | Cucumber mosaic virus |
| CNR | Crinkler & necrosis |
| Col | Columbia |
| COR | Coronatine |
| COX5 | Cytochrome oxidase subunit v |
| CP | Coat protein |
| CRISPR | Clustered regularly interspaced short palindromic repeats |
| CSEPs | Candidate secreted effector protein |
| CT | P-coumaroyl tyramine |
| CWDE | Cell wall degrading enzymes |
| D | Aspartate |
| DAMPs | Damage associated molecular patterns |
| DCL | DICER-like |
| DGE | Differential gene expression |
| DHAR | Dehydroascorbate reductase |
| DMT | DNA methyl transferase |
| DORN1 | Does not respond to nucleotide 1 |

| | |
|---|---|
| DRBs | Double stranded RNA binding proteins |
| DRE | Dehydration responsive element |
| dsDNA | Double stranded deoxyribonucleic acid |
| dsRNA | Double stranded Ribonucleic acid |
| DTI | Danger triggered immunity |
| eATP | Extracellular ATP |
| EBL | Epibrassinolide |
| ECC | *Erwinia carotovora* |
| ECM | Extra cellular matrix |
| ECS | Endocytosis cell signaling |
| EDS 1 | Enhanced disease susceptibility 1 |
| EF | Elongation factor |
| EFR | EF-Tu receptor |
| EF-Tu | Elongation factor Tu |
| EHC | Encasement of the haustorial complex |
| EHM | Extra haustorial membrane |
| EIX | Ethylene inducing xylanase |
| ELISA | Enzyme-linked immunosorbent assay |
| eLRR | Extra cytoplasmic leucine rich repeat |
| EPD | Eukaryotic promoter domain |
| EPSs | Extracellular polysaccharides |
| ER | Endoplasmic reticulum |
| ERE | Ethylene responsive elements |
| EREBP | Ethylene responsive element binding protein |
| ERF | Ethylene response factor |
| ESI | Electrospray ionization |
| ESTs | Expressed sequence tags |
| ET | Ethylene |
| ETI | Effector triggered immunity |
| ETR2 | Ethylene resistant 2 |
| ETS | Effector-triggered susceptibility |
| FISH | Fluorescence in-situ hybridization |
| FLAK | Phenylalanine, leucine, alanine, lysine |
| Flg | Flagellin |
| FLS 2 | Flagellin sensing 2 |
| FMs | Functional markers |
| FT | Feruloyl tyramine |
| GA | Gibberellic acids |
| GC-MS | Gas chromatography mass spectrometry |
| GE | Glucan elicitor |
| GEBP | GE-binding protein |
| GM | Genetically modified |
| GNA | *Galanthus nivalis* agglutinin |
| GR | Glutathine reductase |
| GST | Glutathione s-transferase |

| | |
|---|---|
| HCN | Hydrogen cynide |
| HCRSV | Hibiscus chlorotic ringspot carmovirus |
| hc-siRNAs | Hetrochromatic small interfering RNAs |
| HDA19 | Histone deacetylase 19 |
| HeMV | Hempane mosaic virus |
| HEN1 | HUA ENHANCER |
| HGA | Homogalactouronam |
| HMGR2 | 3-hydroxy-3-methylglutaryl CoA reductase 2 |
| HPLC | High performance liquid chromatography |
| HR | Hypersensitive response |
| hrc | HR and conserved |
| hrp | Hypersensitive reaction and pathogenicity |
| HSN | Host specific necrotroph |
| HSPs | Heat shock proteins |
| HSTs | Host specific toxins |
| HVMPK4 | *Hordeum vulgare* signaling protein MAP kinase 4 |
| HYL1 | Hyponastic leaves 1 |
| IAA26 | Indole-3-acetic acid transcription factor |
| ICAT | Isotope coded affinity tag |
| IEF | Isoelectric focusing |
| IP-ELISA | Immune virus particle-ELISA |
| IPG | Immobilized pH gradient |
| IPP | Isopentenyl diphosphate |
| IPT | Isopentyl transferase |
| ISR | Induced systemic resistance |
| ITRAQ | Isobarric tagged for relative and absolute quantitation |
| JA | Jasmonic acid |
| JARE | Jasmonic acid responsive element |
| JS | Justamembrane |
| LC | Liquid chromatography |
| LC-MS/MS | Liquid chromatography-tandem mass spectrometry |
| L-DOPA | L-3,4-dihydoxyphenylalanine |
| LEA | Late embrogenesis abundant |
| LecRK | Lectin receptor kinase |
| Ler | Landsberg erecta |
| LGD1 | Lagging growth development 1 |
| LIR | Localised induced responses |
| LOS | Lipo-oligosaccharide |
| LPS | Lipopolysachharide |
| LRK | LRR kinase |
| LRR | Leucine rich repeat |
| lsiRNAs | Long small interfering RNAs |
| LTP | Lipid transfer protein |
| LYK3 | LysM receptor like kinase 3 |
| LZNBS-LRR | Leucine zipper nucleotide binding site leucine rich repeat |

| | |
|---|---|
| m/z | Mass to charge ratio |
| MALDI | Matrix assisted laser desorption/ionisation |
| MAMPs | Microbe associated molecular patterns |
| MAPK | Mitogen activated protein kinase |
| MCPs | Methyl accepting chemotaxis proteins |
| MDP | Muramyl dipeptide |
| MeJA | Methyl jasmonate |
| MIMPs | Microbe induced molecular patterns |
| miRNA | MicroRNA |
| MKK | MAP kinase kinase |
| MKS | MPK4 substrate |
| MLA 10 | Mildew resistance locus A10 |
| MoAGOs | M. oryzae genome encoded AGOs |
| MoDCL1 | M. oryzae genome encoded DCL1 |
| MoDCL2 | M. oryzae genome encoded DCL2 |
| MPs | Movement proteins |
| MPSS | Massively parallel signature sequencing |
| MS | Mass spectrometry |
| MSUD | Meiotic silencing of unpaired DNA |
| MTA | 5′-methylthyoadenosine |
| MTI | MAMP triggered immunity |
| MTI | Microbial associated molecular pattern(MAMP) triggered immunity |
| MudPIT | Multidimensional protein identification technology |
| NAG | N-acetylglucosamine |
| NAM | N-acetyl muramic |
| NAMP | Nematode associated molecular pattern |
| NASBA | Nucleic acid sequence based amplification |
| nat-siRNAs | Natural antisense transcript-derived small interfering RNAs |
| NBS | Nucleotide binding site |
| NDPR | Non-pathogen derived resistance |
| NDR 1 | Non race specific disease resistance 1 |
| NEP1 | Necrosis and ethylene inducing protein 1 |
| NF | Nodulation factor |
| NFP | NOD factor perception |
| NGS | Next generation sequencing |
| NLPs | Necrosis and ethylene inducing peptide 1 like proteins |
| NLPs | Nep L like proteins |
| NLS | Nuclear localization signal |
| NO | Nitric oxide |
| NOD | Nucleotide binding oligomerization domain |
| NPAA | Nonprotein amino acids |
| NRG1 | N-requirement gene |
| NtMKP1 | Tobacco MAP kinase phosphatase 1 |
| nTNL | Non TIR-NBS-LRR |
| $O_2$ | Oxygen |

| | |
|---|---|
| OBF | Octopine synthase element binding factor |
| Obpv | Obuda pepper virus |
| OG | Oligogalacturonides |
| ORCAs | Octadecanoid responsive catharanthus APETALA2 domain proteins |
| Osa | *Oryza sativa* |
| osNramp6 | osa-miRNA negative regulation of natural resistance associated macrophage protein 6 |
| PAB | Plant associated bacteria |
| PAD3 | Phytoalexin deficient 3 |
| PAL | Phenylalanine ammonia lyase |
| PAMPs | Pathogen associated molecular pattern |
| PCD | Programmed cell death |
| PCR | Polymerase chain reaction |
| PD | Plasmodesmata |
| PDR | Pathogen derived resistance |
| Pel | Pectate lyase |
| PEN | Penetration genes |
| PepMV | Pepino mosaic virus |
| PEST | Pro-Glu-Ser-Thr |
| PG | Polygalactouronases |
| PGIP | Polygalactouronase inhibiting protein |
| PGN | Peptidoglycan |
| pI | Isoelectric point |
| PKs | Protein kinases |
| PLRV | Potato leafroll virus |
| PMEs | Pectin methyl esterases |
| PNPs | Plant natriuretic peptides |
| Pop | *Pseudomonas* outer protein |
| PPO | Poly phenol oxidase |
| PPP | Pentose phosphate pathway |
| PPV | Plum pox potyvirus |
| PR | Pathogenesis related |
| PR1 | Pathogenesis related elements |
| pre-miRNA | Precursor miRNA |
| pri-miRNA | Primary-miRNA |
| PRLs | PR like proteins |
| PRRs | Pathogen recognition receptors |
| PRRs | Pattern recognition receptors |
| PRSV | Papaya ring spot virus |
| PS I | Photosystem I |
| PS II | Photosystem II |
| PSE1 | Penetration specific effector 1 |
| PstDC3000 | Pathovar tomato strain DC3000 |
| PTA-ELISA | Plate trapped antigen-ELISA |
| PTGS | Post transcriptional gene silencing |

| | |
|---|---|
| PTGS | Post translational gene silencing |
| PTI | PAMP triggered immunity |
| PTMs | Post translational modification |
| PVX | Potato virus X |
| PVY | Potato virus Y |
| PX | Peroxiredoxins |
| QAs | Quinolizidine alkaloids |
| qPCR | Real time quantitative PCR |
| QS | Quorum sensing |
| QTL | Quantitative trait loci |
| R | Resistance |
| RALF | Rapid alkalisation factor |
| ra-siRNAs | Repeat associated small interfering RNAs |
| RdDM | RNA-directedDNAmethylation |
| RDR | RNA dependent RNA polymerase |
| RDV | Rice dwarf virus |
| REL | Reticuloendotheliosis |
| REn | Replication enhancer |
| RenSeq | R gene enrichment and sequencing |
| RGC2 | Resistance gene candidate 2 |
| RIP | Repeat induced point |
| RISC | RNA induced silencing complex |
| RLCKs | Receptor like cytoplasmic kinases |
| RLK | Receptor like kinase |
| RLP | Receptor like protein |
| RMs | Random markers |
| RNAi | RNA interference |
| RNS | Reactive nitrogen species |
| ROMT | Resveratrol-o-methyltransferase |
| ROS | Reactive oxygen species |
| RP | Reverse phase |
| RPA | Reverse phase protein microarray |
| RPW8 | Resistance to powdery mildew 8 |
| Rsp1 | Repetitive secreted protein 1 |
| RSS | RNA silencing suppressors |
| RTD | Read through domain |
| RTP | Read through protein |
| RT-PCR | Reverse transcriptase-PCR |
| RUBISCO | Ribulose-1,5-biphosphate carboxylase oxygenase |
| RYMV | Rice yellow mottle virus |
| SA | Salicylic acid |
| SAGE | Serial analysis of gene expression |
| SAM | S-adenosyl methionine |
| SAR | Systemic acquired resistance |
| SARE | Salicylic acid responsive elements |

| | |
|---|---|
| SARE | SA-responsive element |
| SCF | SKP1-cullin-f-box protein |
| SCX | Strong cation exchange |
| SDS-PAGE | SDS- polyacrylamide gel electrophoresis |
| SE | Sieve element |
| See1 | Seedling efficient effector 1 |
| SEL | Size exclusion limit |
| SERK3 | Somatic embryo receptor kinase 3 |
| SIB1 | Sigma factor binding protein 1 |
| SIPK | Salicylic acid protein kinase |
| SIS | Sex induced silencing |
| SMV | Soybean mosaic virus |
| SNARE | Soluble N-Ethylmaleimide-sensitive factor attachment protein receptor |
| SNPs | Single-nucleotide polymorphisms |
| SOD | Superoxide dismutase |
| sRNAs | Small RNAs |
| ssDNA | Single stranded deoxyribonucleic acid |
| SSEM | Serologically specific electron microscopy |
| SSH | Suppression subtractive hybridization |
| SSITL | *Sclerotinia sclerotiorum* integrin like |
| SSPs | Small secretory proteins |
| SSR | Simple sequence tags |
| ssRNA | Single stranded ribonucleic acid |
| SSRs | Simple sequence repeats |
| STAND | Signal transduction ATPases with numerous domains |
| STB | Septoria tritici blotch |
| STS | Stilbone synthase gene |
| SYMRK | Symbiosis receptor kinase |
| ta-siRNAs | Trans-acting small interfering RNAs |
| TBP | TATA-box -binding protein |
| TCNL | TIR-CC-NBS-LRR |
| TCV | Turnip crinkle virus |
| TFs | Transcription factors |
| TGB | Triple gene block |
| TGS | Transcriptional gene silencing |
| THI2.1 | Thionin 2.1 |
| TIPK | Trichoderma induced MAPK |
| TIR | Toll interleukin 1 receptor |
| TLR 4 | Toll like receptor 4 |
| TMV | Tobacco mosaic virus |
| TNL | TIR-NBS-LRR |
| TOF-MS | Time of flight mass spectrometry |
| TrD | Transmembrane domain |
| tRNA | Transfer RNA |

| | |
|---|---|
| TSWV | Tomato spotted wilt virus |
| TTSS | Type three secretion system |
| TuMP | Turnip mosaic virus |
| TVMV | Tobacco vein molting virus |
| TYLCD | Tomato yellow leaf curl disease |
| VIP1 | VirE2 interacting protein 1 |
| VOCs | Volatile organic compounds |
| VRC | Viral replication complex |
| vRNP | Viral ribonucleoprotein complex |
| W | Tryptophan |
| WAK1 | Wall associated kinase 1 |
| WIPK | Wound induced protein kinase |
| Xop | Xanthomonas outer protein |

# 1 *Arabidopsis thaliana* as a Model Organism to Study Plant-Pathogen Interactions

Shachi Agrawal

**Abstract**

*Arabidopsis thaliana* (*a crucifer*) provides a model system in every discipline of plant sciences including plant pathology with a varied array of molecular and genetic resources and biological information. Members of crucifer are widely distributed geographically and are well adapted to various plant pathogens such as fungi, bacteria, viruses, and nematodes. Besides small plant size, short life cycle, small genome size, availability of whole genome sequence, and easy genetic and mutational analysis, its response to the pathogen attack in a similar fashion as other higher plant species and an extensive collection of mutants available to determine defense pathway are the characteristics, which identify this plant as an indispensable research model in plant-pathogen interaction studies. This chapter mainly focuses on various existing model pathosystems of *Arabidopsis* with viral, bacterial, and fungal pathogens including an outlook on how this knowledge can be translated from *Arabidopsis*-pathogen model system to other crop plants. A general and brief overview of plant-pathogen interactions and how *A. thaliana* recognize and respond to pathogens is also portrayed.

**Keywords**

Effector molecules · Hypersensitive response (HR) · Plant defense · Plant defensin gene · PR proteins · Resistance genes · Signal molecules · Systemic acquired resistance (SAR)

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## 1.1 Introduction

When a pathogen attacks a plant, either a pathogen can proliferate and can cause development of disease or the plant can resist the pathogen by means of active or passive form of resistance. During resistance, plants recognize a race-specific avirulence determinant produced by the pathogen (Keen 1990; Scofield et al. 1996; Tang et al. 1996); defense mechanisms are activated leading to hypersensitive response (HR) (Matthews 1991). At the same time, expressions of pathogenesis-related (PR) proteins as well as plant defensins are induced due to gene-for-gene interactions and rapid localized cell death (Narasimhan et al. 2001; Asano et al. 2012). Signaling molecules, salicylic acid (SA), jasmonic acid (JA), ethylene (ET), and reactive oxygen species (ROS) are directly involved in plant defense against pathogens (Clarke et al. 2000; Kunkel and Brooks 2002; Hossain et al. 2007; Asano et al. 2012). There is rich information on plant-pathogen interaction on many species. Advanced molecular tools are also accessible that can be used to study the function and evolution of genes that are important for plant defense such as those that control responses to wide range of pathogens. However, studies in molecular plant pathology require large initial investments in molecular technologies. It is cost-effective since these investments are shared among multiple laboratories by means of publications, bioinformatic tools, and databases such as TAIR. Moreover, researchers gain in-depth biological understanding when they compare and match previous studies from a research community that shares the tools and resources of model organisms. Although, it is imperative to study individual plant-pathogen interactions at species level to gain better knowledge. But, at the same time, *A. thaliana* serves as a model system to answer many basic questions related to plant-pathogen interaction due to availability of complete genome sequence and having a small genome size together with the extensive collection of new mutants and germplasm as well as the presence of specialized transformation techniques, its rapid growth, can be handled easily in the laboratory conditions, mutagenesis can also be done easily and the possibility of using microarrays for gene expression analysis. *Arabidopsis* is susceptible to only a limited number of pathogens including viruses, bacteria, fungi, nematodes, and insect pests, and it responds to the pathogen attack in a similar fashion to those of other higher plant species.

*A. thaliana* (L.) Heynh. is an annual flowering plant that belongs to mustard family (Cruciferae or Brassicaceae). It is a native of Eurasia, which has a broad natural distribution throughout Europe, Asia, and North America. Of late it has been introduced and naturalized worldwide. It is speculated that its spread was facilitated by the expansion of agriculture (Francois et al. 2008). *A. thaliana* is considered as a weed as it grows in open or recently disturbed habitats. *Arabidopsis* shows extensive natural variation for different developmental, abiotic, and biotic stress resistance traits (Koornneef et al. 2004; Alonso-Blanco et al. 2009; Atwell et al. 2010). Till date, over 750 different ecotypes (accessions) of *A. thaliana* have been collected from natural populations that are available for experimental analyses. The most commonly used ecotypes of *Arabidopsis* for genetic and molecular studies are Columbia (Col) and Landsberg erecta (L*er*). The entire life cycle of *A. thaliana* is completed in 6 weeks, which includes seed germination, rosette formation, bolting,

**Fig. 1.1** An *Arabidopsis* plant grown under laboratory condition

flowering, and maturation of seeds. The plant is a small-sized herb with overall length of around 15–20 cm; leaves are 1.5–5 cm long and 2–10 mm broad. Flowers (2 mm length and 3 mm diameter) undergo self-pollination but can be crossed manually. The fruit is called a silique (5–20 mm long) that contains 20–30 seeds. On germination, the seed develops into a rosette plant (2–10 cm diameter), wherein the whorls of leaves are covered with trichomes (Fig. 1.1). Under laboratory conditions *Arabidopsis* can be grown easily in petri plates, pots, or hydroponics, either under fluorescent lights or in a greenhouse. Inflorescence is a corymb that appears as a result of bolting after 3 weeks of planting. Several hundred siliques are produced per plant, which account for more than 5000 total seeds. The plant has a single primary root that grows vertically downward and produces smaller lateral roots that are easy to study in culture.

## 1.2 Plant-Pathogen Interactions

An array of pathogens including fungi, bacteria, and viruses attack the plant kingdom. Different strategies have been devised by different pathogens to invade, feed on, and reproduce in the host plants. Pathogens can be broadly classified as biotrophs and necrotrophs based upon the strategy used by them to invade and infect a plant (Oliver and Ipcho 2004). Biotrophic pathogens are those that require a living host tissue for its growth and reproduction. In some cases wherein the tissue dies in the later stages of the infection, the pathogens are classified as hemibiotrophs. On the

contrary, necrotrophic pathogens kill the host tissue as soon as they infect it and then grow and feed on the dead tissue. Viruses are classified as biotrophic pathogens, whereas bacteria and fungi follow both biotrophic as well as necrotrophic strategies of invasion. Plants respond to different kinds of pathogens differently. Pathogens can further be classified as those with different primary target tissues encountering different environmental conditions. Those pathogens that target the green, photosynthesizing, and assimilate-producing source tissues like leaves will encounter different kinds of defense responses in comparison to pathogens infecting the assimilate-importing tissue such as roots, flowers, and sink leaves (Berger et al. 2007).

Plant defense mechanism against pathogens can be either preformed (primary) or induced (secondary). The first and foremost step required for the activation of defense response is to recognize the presence of microorganisms. Elicitors are molecules that at very low concentrations induce plant defense response (Thakur and Sohal 2013). Recognition of microorganism-derived elicitors initiates the basal resistance in plants. This defense response involves activation of ion fluxes, phosphorylation/dephosphorylation of proteins by protein kinases and phosphatases, and production of signaling molecules such as adapter proteins, salicylic acid, jasmonic acid, ethylene, reactive oxygen species, and nitric oxide. These steps further initiate an array of signaling that leads to the regulation of expression of defense-related genes and the induction of defense responses. These responses include cell wall strengthening, accumulation of phytoalexins and pathogenesis-related (PR) proteins, and localized programmed cell death (PCD) (McDowell and Dangl 2000; Dangl and Jones 2001; Garcia-Brugger et al. 2006).

Plants also possess an innate immune system that perceives the presence of pathogens by recognition of molecules known as microbe- or pathogen-associated molecular patterns (MAMPs or PAMPs, respectively) or by sensing effector proteins secreted by the host during plant-pathogen interactions. Early interactions between PAMPs/MAMPs and cell surface receptors (pathogen recognition receptors or PRRs) lead to appropriate defenses by activating multicomponent and multilayered responses. The establishment of defense is triggered by several pathways that can involve $Ca^{2+}$ influx, generation of reactive oxygen and nitrogen species (ROS and RNS, respectively), and synthesis of phytohormones such as jasmonic acid (JA), salicylic acid (SA), and ethylene (ET), which act as signal molecules (Pieterse et al. 2009). Plant immunity may be described at two levels (Jones and Dangl 2006). The first one involves cell surface pattern recognition receptors (PRRs) to detect pathogen-associated molecular patterns (PAMPs) and initiate PAMP-triggered immunity (PTI). The second involves nucleotide-binding leucine-rich repeat (NB-LRR) proteins, encoded by resistance (*R*) genes, which sense pathogen effectors and elicit a potent immune response called effector-triggered immunity (ETI). ETI is faster, longer, and stronger than PTI and usually leads to a local cell death, the hypersensitive response (HR), which stops the spread of the pathogen (Jones and Dangl 2006). In some cases, pathogens can evade such recognitions also and suppress host immunity with effectors, causing effector-triggered susceptibility

(ETS). R proteins recognize some effectors that enable the pathogen to overcome PTI, and the effectors are thus termed an avirulence (Avr) protein (Jones and Dangl 2006).

As per our current understanding, virulence of the virulent pathogenesis is contributed by the production of effector molecules which thereby suppress plant defense, and thus the compatible interactions allow the spread of the pathogen in the susceptible plant (Jones and Dangl 2006). Herein the pathogen proliferates at a rate in which the plant defense could not keep pace with that subsequently leads to the development of disease and necrosis. On the other hand, in resistant plants, the specific resistance is governed by the recognition of the activity of pathogen effector molecules (race-specific avirulence determinant) by plant receptor proteins (Keen 1990; Scofield et al. 1996; Tang et al. 1996; Berger et al. 2007). Hence, these incompatible interactions prevent the pathogen from spreading and impart resistance to the plant. The disease resistance "R" genes encode the microbe recognizing plant receptors. Those pathogens that cannot establish themselves in the host plant are called as avirulent strains of plant pathogens, and their early recognition combined with fast activation of plant defense mechanisms results in the inducible defense system (Jones and Dangl 2006). Moreover, the recognition of the avirulent strain determinant activates a hypersensitive response (HR) that is characterized by localized PCD resulting in small necrotic lesions that efficiently restrict the spread of biotrophic pathogens (Heath 2000; Narasimhan et al. 2001). In addition, plant defensins (PDF1.1, PDF1.2) mRNAs are expressed in response to gene for gene interaction (Narasimhan et al. 2001). As discussed earlier, various signaling molecules like jasmonic acid, salicylic acid, ethylene, and reactive oxygen species (ROS) are directly involved in such inducible defense systems (Clarke et al. 2000; Kunkel and Brooks 2002; Hossain et al. 2007). The jasmonate/ethylene signaling pathway seems to be the most important mechanism in defending against necrotrophic pathogens. On the other hand, in order to combat against the biotrophic pathogens, plants recruit the salicylic acid-dependent responses (Thomma et al. 2001).

## 1.3 How *Arabidopsis thaliana* Recognize and Respond to Pathogens?

In nature, plants are exposed to a large number of pathogens, but somehow they are susceptible to only a few of them. This may be due to the presence of different defense mechanisms exhibited by the plants (Nimchuk et al. 2003; Jones and Takemoto 2004). The disease resistance (*R*) genes that are involved in pathogen recognition show excessive polymorphism. This polymorphism has been speculated as a cause for plant resistance. In monoculture, loss of R gene polymorphism results in reduced resistance and increased susceptibility (Stahl and Bishop 2000). *Arabidopsis* is prone to infection by pathogens that includes viruses, bacteria, fungi, nematodes, and insects. As the mode of response to the pathogen attack is highly

conserved in higher plant species, study of *Arabidopsis*-pathogen interactions have greatly helped the scientists to understand the molecular and cellular basis of host-pathogen interactions, disease resistance, and pathogen virulence (Andargie and Li 2016).

As stated earlier, R genes are important for parasite recognition and initiation of defense mechanism. A total of 150 different R genes have been identified in *Arabidopsis* genome that are located unevenly on chromosomes with 49, 2, 16, 28, and 55 R gene loci on chromosome number 1, 2, 3, 4, and 5, respectively (*Arabidopsis*-Genome-Initiative 2000). These R genes encode for proteins that contain nucleotide-binding (NB) domain(s) that binds to ATP or GTP along with a carboxy-terminal leucine-rich repeat (LRR) domain (S) that facilitate protein-protein interactions and ligand binding. They are further classified as those that contain toll interleukin 1 receptor domain (TIR) or coiled-coil (CC) domain at their amino terminal. Thus, broadly they can be classified as TIR-NB-LRR and CC-NB-LRR. *Arabidopsis* genome contains 85 TIR-NB-LRR resistance genes at 64 loci and 36 CC-NB-LRR resistance genes at 30 loci (*Arabidopsis*-Genome-Initiative 2000). Some of these R genes carry additional domains also, like WRKY transcription factor domain and protein kinase domain that have also been implicated in plant defense.

Studies were carried out to compare the defense mechanisms in plants and animals. Nitric oxide production seems to be a common response in both plants and mammals in conditions of biotic stress. But distinct homologue of nitric oxide synthase gene was not found in *Arabidopsis*. REL (reticuloendotheliosis) domain transcription factors or similar proteins that are involved in innate immunity in both *Drosophila* and mammals or their homologs were not detected in *Arabidopsis thaliana*. Moreover, no homologues were detected for genes like classical caspases, bcl2/ced9, and baculovirus p35 that are involved in apoptosis regulation in animal cells; however, eight homologues of metacaspase family protein and 36 cysteine proteases were found in *Arabidopsis* (*Arabidopsis*-Genome-Initiative 2000; Uren et al. 2000). The production of reactive oxygen intermediates is a primary response that is common to both plant and animal during pathogen recognition. This process involves transfer of electrons across the plasma membrane in mitochondria to make superoxide by a specialized respiratory burst oxidase. In mammals, gp91 is the subunit of NADH oxidase that catalyzes the final step of electron transfer to molecular oxygen ($O_2$), resulting in the generation of superoxide ion ($O_2^-$) (Yu et al. 1998). The *Arabidopsis* genome has eight functional homologues of *gp91*. These homologues are called *Atrboh* genes and have been implicated in plant defense response (Torres et al. 2002). In mammals, gp91 activity requires the action of Rac proteins, but no Rac or Ras proteins are found in *Arabidopsis*; however, a large family of rop genes that are related to G-proteins are present and may carry the same function. The various pathogens of *Arabidopsis thaliana*, gene associated with natural variation of response to those pathogens and their molecular functions, are summarized in Table 1.1.

**Table 1.1** Various pathogens of *Arabidopsis thaliana* along with gene associated with natural variation of response to pathogen interactions and their molecular functions (Roux and Bergelson 2016)

| Pathogens of *Arabidopsis* | Associated gene locus | Class of the associated gene |
|---|---|---|
| **Viruses** | | |
| *Turnip crinkle virus* (TCV) | HRT | CC-NBS-LRR protein |
| *Cucumber mosaic virus* (CMV) | RCY1 | CC-NBS-LRR protein |
| *Tobacco ringspot virus* (TRSV) | TTR1 | TIR-NBS-LRR protein |
| *Tobacco etch virus* (TEV) | RTM1 | Jacalin-like lectin protein |
| | RTM2 | Small heat shock-like protein |
| | RTM3 | MATH domain-containing protein |
| *Plum pox virus* (PPV) | RTM1 | Jacalin-like lectin protein |
| | RTM2 | Small heat shock-like protein |
| | RTM3, rwm1/rpv1 | MATH domain-containing protein |
| | | Nucleus-encoded chloroplast phosphoglycerate kinase |
| *Lettuce mosaic virus* (LMV) | RTM1 | Jacalin-like lectin protein |
| | RTM2 | Small heat shock-like protein |
| | RTM3 | MATH domain-containing protein |
| *Plantago asiatica mosaic virus* (PAMV) | JAX1 | Jacalin-like lectin protein |
| *Watermelon mosaic virus* (WMV) | rwm1/rpv1 | Nucleus-encoded chloroplast phosphoglycerate kinase |
| **Bacteria** | | |
| *Pseudomonas syringae* | RPM1/RPS3 | CC-NBS-LRR protein |
| | RPS2 | CC-NBS-LRR protein |
| | RPS5 | CC-NBS-LRR protein |
| | RPS4 | TIR-NBS-LRR protein |
| | RRS1 | TIR-NBS-LRR WRKY protein |
| | ACD6 | Ankyrin-repeat transmembrane protein |
| *Xanthomonas campestris* | RPS4 | TIR-NBS-LRR protein |
| | RRS1 | TIR-NBS-LRR WRKY protein |
| | RKS1 | A typical kinase |
| | AT5G22540 | Protein of unknown function |
| *Ralstonia solanacearum* | RPS4 | TIR-NBS-LRR protein |
| | RRS1 | TIR-NBS-LRR WRKY protein |
| | ERECTA | LRR receptor-like kinase |
| **Fungi** | | |
| Elicitor from *Sclerotinia sclerotiorum* | RLP30 | Receptor-like protein |
| *Botrytis cinerea* | RLP30 | Receptor-like protein |
| | EGM1 | Receptor-like kinase |
| | EGM2 | Receptor-like kinase |
| | RLM3 | TIR-NB protein |

(continued)

**Table 1.1** (continued)

| Pathogens of *Arabidopsis* | Associated gene locus | Class of the associated gene |
|---|---|---|
| *Fusarium oxysporum* | RFO1 | Wall-associated receptor-like kinase |
| | RFO2 | Receptor-like protein |
| | RFO3 | Receptor-like kinase |
| *Alternaria brassicicola* | RLM3 | TIR-NB protein |
| *Alternaria brassicae* | RLM3 | TIR-NB protein |
| *Colletotrichum higginsianum* | RPS4 | TIR-NBS-LRR protein |
| | RRS1 | TIR-NBS-LRR WRKY protein |
| Oomycetes | | |
| *Albugos candida* | RAC1 | TIR-NBS-LRR protein |

## 1.4 *Arabidopsis thaliana*: An Important Model Host for Studying Plant-Pathogen Interactions

*A. thaliana* is an important model host for studying plant-pathogen interactions due to several reasons as described earlier. *Arabidopsis* is susceptible to only a limited number of pathogens including viruses, bacteria, fungi, nematodes, and insect pests. Diseases resulting from these pathogens have been reported in the wild (Holub et al. 1994, 1995; Tsuji and Somerville 1992) suggesting both the pathogen and the host share an ecological niche, and when the appropriate environmental conditions are present, disease can occur. Diseases have also been observed in a laboratory setting where the host is deliberately exposed to the pathogen. Regardless of the setting, nature, or the laboratory, *Arabidopsis* responds to the pathogen attack in a similar fashion as other higher plant species when exposed to viral, prokaryotic, or eukaryotic pathogens (Andargie and Li 2016). Since the 1990s till today, several plants have been recognized as model systems for plant-pathogen interactions such as tobacco, tomato, etc., but *A. thaliana* has been used extensively as a model plant to have an overview of the plant-pathogen interactions with a wide variety of pathogens. The *A. thaliana* genetic system is significantly more tractable than those of the other plant species, which were hampered by long generation times and large, polyploid, or repetitive genomes. Agriculturally important crucifers such as *Brassica napus*, *Brassica rapa* (oilseed rape, canola), *B. oleracea*, *Brassica* spp., European cabbage, cauliflower, Chinese cabbage, and radish (*Raphanus* spp.) are the closest relatives of *Arabidopsis*, so all the informations available can be useful for studying plant-pathogen interactions in these related spp. that are economically important crops. But molecular studies can be more complex in these spp. since they are mostly polyploids.

Besides, *A. thaliana* exhibits all of the major kinds of defense responses described in other plants. Furthermore, a large number of virulent and avirulent bacterial, fungal, and viral pathogens of *A. thaliana* have been deciphered (Glazebrook et al. 1997). Mutants defective in almost every aspect of plant growth and development have been identified and studied by the various research groups over the world. Novel insights into events subsequent to pathogen recognition in *A. thaliana* have

been obtained from mutants altered in defense (Buell 1998). Several mutant groups in *A. thaliana* exist today: lesion mimic mutants, phytoalexin mutants, as well as enhanced susceptibility and resistance mutants. With the variety of mutants available, it is possible to determine which defense pathways are activated during pathogen attack and what leads to the subsequent resistance or susceptibility. As research progresses, the different mutants will be linked to specific genes finally leading to a better understanding of the various genes involved in plant response pathways (Glazebrook et al. 1997).

## 1.5 *A. thaliana*-Pathogen Interactions

*Arabidopsis* has been reported as a susceptible host to a range of pathogens and resistant to other pathogens. The findings related to defense mechanism in *Arabidopsis* have been successfully implemented in many model systems, which have been developed to better understand interactions between plants and pathogens. The primary response of *Arabidopsis* includes the perception of pathogens by cell surface pattern recognition receptors (PRRs) and is referred to as PAMP-triggered immunity (PTI). Activation of FLS2 and EFR triggers MAPK signaling pathway that activates defense genes for synthesis of antimicrobial compounds. *Arabidopsis* possess specific intracellular surveillance proteins (R proteins) to monitor the presence of pathogen virulence proteins. This ETI occurs with localized programmed cell death to arrest pathogen growth, resulting in cultivar-specific disease resistance.

### 1.5.1 *Arabidopsis*-Virus Interactions

Viral infections and their spread throughout a plant require numerous interactions between the host and the virus. Systemic viral infections in plants are complex processes that require compatible virus-host interactions in multiple tissues. These interactions include viral genome replication in the cytoplasm of the initially infected cells, cell-to-cell movement toward neighboring tissues, long-distance movement through the vascular tissue, phloem unloading, and cell-to-cell movement in non-inoculated systemic tissues (Carrington et al. 1996). Incompatibilities between virus and host factors at any of these stages could therefore lead to restrictions and delay establishment of a systemic infection. The utility of *Arabidopsis* as a model system has not gone unnoticed, and several viruses previously found to be pathogenic on crucifers have also been found to infect *Arabidopsis*. This model organism has proven to be useful to understand the relationship between the host plant and the virus replication and movement processes (Kunkel 1996; Yoshii et al. 1998). Susceptible interactions between plants and viruses can result in a variety of visible symptoms ranging from mild stunting to overall necrosis.

Although plant viruses are among the least genetically complex pathogens, they use a variety of strategies to suppress or bypass host defense and infect susceptible